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In Re: Application of ) Art Unit:  
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Kathleen L. Hannan )  
Serial No.: ) Examiner:  
Filed: )  
For: Brainstem and Limbic )  
Disorder (BALD) )

This application is a continuation in part and division of application number 09/503,656 filed February 04, 2000 entitled Brainstem and Limbic Disorder (BALD).

1. Field of the invention.

This invention relates to neuro-toxicity and disorders of the central nervous system. Specifically disorders of the brain stem and limbic system of the brain are localized in diagnosis.

The acronym BALD is used to refer to the brainstem and lymbic system disorder in this specification.

2. Background

In order to fully understand the nature of the invention described herein it is necessary to briefly describe the anatomy, physiology, biochemistry, and pathology that is related to the present invention.

Related anatomy:

The brain stem consists of the medulla oblongata, pons, and midbrain or mesencephalon. The lower end of the brain stem is a



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1 continuation of the spinal cord. Within the medulla are several  
2 structures that relate to the present invention. There is a  
3 conduction pathway for motor and sensory impulses between the brain  
4 and spinal cord called the reticular formation. Within the  
5 reticular formation of the medulla are three vital reflex centers:  
6 1) the cardiac center which regulates the rate of heartbeat and  
7 force of contraction; 2) the medullary rhythmicity area which  
8 adjusts the basic rhythm of breathing; and 3) the vasoconstrictor  
9 center which regulates the diameter of blood vessels.

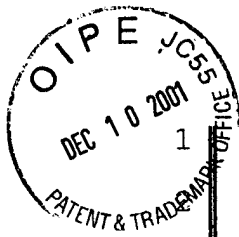
10 Associated with the brain stem are twelve cranial nerves  
11 designated by name and roman numerals I to XII. The so called  
12 autonomic nervous system consists of both parasympathetic and  
13 sympathetic nerve fibers. The sympathetic nerve fibers arise from  
14 various segments of the spinal cord and the parasympathetic nerve  
15 fibers arise from the brain stem. The so called cranial nerves  
16 taken as a group comprise the parasympathetic nervous system.

17 The limbic system is a wishbone-shaped group of structures  
18 that encircles the brain stem.

19 Physiology:

20 The cranial nerves taken as a group regulate the functions of  
21 the organs of the body and the so called endocrine glands. Thus a  
22 disorder of the brain stem could produce pulmonary, cardiac, or  
23 gastric disturbances and symptoms. A disorder of the brain stem  
24 could even produce disturbances and symptoms in virtually any part  
25 of the physical body of a person.

26 The limbic system functions in the emotional aspects of  
27 behavior, especially behavior related to survival. Lesions in the  
28 limbic system result in impairment of memory functions as well as



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1 in emotional disturbances.

2 Biochemistry:

3 The chief source of energy in the cells of the body except of  
4 the so called red blood cells is the result of the breakdown of  
5 glucose. This glucose breakdown is known as glycolysis and results  
6 in the production of pyruvic acid. A series of biochemical  
7 reactions called the citric acid cycle or the tricarboxylic acid  
8 cycle (TCA cycle) breaks down the pyruvic acid into energy  
9 molecules and carbon dioxide. The carbon dioxide is subsequently  
10 expelled from the lungs.

11 Generally the biochemical reactions in energy production are  
12 as follows:

13 Carbohydrate => sugar => pyruvic acid => NAD, and NADH and Carbon  
14 Dioxide

15 The nine known general steps of the TCA cycle are as follows:

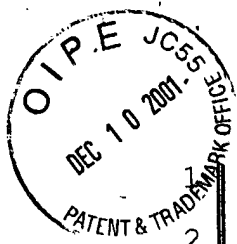
16 Pyruvic acid =>

17 acetyl coenzyme A => Citric acid => cis-aconitic acid => isocitric  
18 acid => NAD => NADH => Succinyl Co A => Succinic acid => Fumaric  
19 acid => Malic acid => Oxaloacetic acid

20 In summary the pyruvic acid is converted in part to acetyl  
21 coenzyme A which is converted in the TCA cycle to carbon dioxide  
22 and energy. The energy produced is stored in molecules of NAD,  
23 NADH, and FAD. NAD is the abbreviation of nicotinamide adenine  
24 dinucleotide. FAD is the abbreviation for flavin adenine  
25 dinucleotide. This NAD, NADH, and FAD are converted to ADP and ATP  
26 then used in various parts of the body in energy production.

27 Pathology:

28 The medulla portion of the brainstem is important in reflexes



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1 for cardiac, vasoconstrictor, and respiratory systems of the body.  
2 Accordingly lesions in the brain stem can and do result in heart  
3 dysfunction, vascular disturbances, and pulmonary disorders.  
4 Lesions in the midbrain part of the brain stem can and do produce  
5 motor nerve dysfunction. Extrapyramidal symptoms are produced by  
6 lesions in the midbrain.

7 Lesions of the limbic system can and do produce abnormal  
8 moods, feelings, emotions, especially the reactions of fear, rage  
9 and emotions related to sexual behavior.

10 Recent clinical observations reveal that a chronic lesion of  
11 the brain stem will also affect the limbic system so that the  
12 patient with the so called BALD syndrome will experience emotional  
13 disturbances as well as physical disorders.

14 One of the discoveries of the present invention is that  
15 lesions of the brain stem and limbic system produce a condition of  
16 hypercalcemia of various parts of the body including the brain stem  
17 and limbic system themselves. It has been found clinically that  
18 this condition of hpyercalcemia results in impairment of the  
19 function of the so called TCA cycle resulting in loss of energy  
20 production in cells of the body.

21 A more detailed description of the background of this  
22 invention follows.

23 The central nervous system is defined as the brain and spinal  
24 cord. Associated with the brain are twelve Cranial Nerves which  
25 enervate specific areas of the body. These cranial nerves are  
26 associated with the brain stem. The brain stem is the area of  
27 the brain where the spinal cord connects with the brain itself. The  
28 limbic system of the brain refers to a ring of structures that form

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1 a border around the brainstem and corpus callosum of the brain.  
2 There are a number of recognized disorders of the central  
3 nervous system. These disorders include seizure disorders such as  
4 epilepsy, vascular disorders producing headache, and degenerative  
5 disorders. CNS degenerative disorders include inherited  
6 degenerative diseases which includes Amyotrophic Lateral Sclerosis  
7 (ALS) and nutritional degenerative disease such as vitamin  
8 deficiencies and alcoholic abuse. Extrapramidal syndromes are  
9 disorders which arise from lesions principally in the basal  
10 ganglia. Parkinsonism is another such degenerative disease which,  
11 like extrapyramidal syndrome, involves movement disorders. There  
12 are also autoimmune disorders such as Myasthenia Gravis which  
13 affect the neuromuscular junction, and other immune disorders  
14 which affect the Myelin insulation of the nerves, or their ability  
15 to produce critical brain chemicals.  
16 It is well known that the brain is vulnerable to injuries. It  
17 is less well known that the deep structures of the brain are more  
18 vulnerable than crush or traction type injuries. Further, the  
19 Nervous System is vulnerable to more than just trauma and acute  
20 infection. The brain demonstrates vulnerability to changes in  
21 levels of glucose, ammonia and other simple molecules. It  
22 demonstrates exquisite responsitivity to deprivation of Oxygen and  
23 glucose. The brain reacts dramatically to solvents used in  
24 mechanical repair areas, to carbon monoxide fumes such as from  
25 propane combustion and to toxins such as phenol and toluene. In  
26 addition, it demonstrates immune reactivity as in the disease  
27 cerebral lupus, to chronic ischemic vascular disease as in as in  
28 diabetes, to autoimmune illness as in certain types of cancers, and



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1 to oxidative stress from liver failure and concomitant excess of  
2 ammonia.

3 The result in the brain of these combinations of causative  
4 conditions, with a final single or ensemble of acute insults is,  
5 both central and peripheral neuropathy as well as eventual muscle  
6 weakness and limb numbness, and pain. Eventually the individual  
7 develops a set of nervous manifestations characterized by  
8 headaches, fatigue, sleep difficulties, heart rate instability, and  
9 gastrointestinal disturbance.

10 The victim of the BALD syndrome described herein develops  
11 symptoms other than at the brain and immune system, such as rashes  
12 from immune problems and infections, chronic multiple fungal  
13 infections, pulmonary, lung and peripheral vascular disease, as  
14 well as premature aging on a cellular level. There can be  
15 increasing autonomic instability, with body temperature  
16 instability, abnormalities of blood flow to organs and skin, and  
17 abnormal salivation and sweating . The diagnostic protocols will  
18 examine all these symptoms.

19 All types of diseases, including the neurological diseases are  
20 more common in the urban environment, where there is concomitant  
21 exposure to industrial and environmental toxic, and polluting  
22 substances. People are more frequently developing extreme  
23 vulnerability to everyday fumes and substances that many people  
24 tolerate. This is being named Multiple Chemical Sensitivity. There  
25 appears to be an increased risk for chronic diseases, including  
26 Diabetes, Asthma, and also the Neurological/Neurotoxic diseases. A  
27 recent study demonstrated that among the black population, babies  
28 who were conceived and had embryonic; fetal life in urban America

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1 are at more than twice the risk to be stillborn. This is of source  
2 neurological death in utero. It is clearly related to the multiple  
3 sources for neurotoxicity in our urban environment.

4 A core concept of this invention is that there can be chronic  
5 inflammation in neuronal tissue, and loss of adequate Oxidation/  
6 energy from oxidative metabolism in critical neural control centers  
7 in the brain. Further, this invention seeks to demonstrate that  
8 immune abnormalities, degradation of connecting axons in the brain,  
9 chronic infection especially by fungi and sequestered neuro toxins.  
10 This leads eventually to the development of autoantibodies, and  
11 chronic electrical instability in neural circuits. Prebirth  
12 children would be of course at greatest risk for this type of  
13 pathological process. Crib death has just been shown to be the  
14 result of fungi acting on the fire retardant in the crib's bedding,  
15 causing the production of three types of poison  
16 gas. This causes asphyxia in small babies, whose nervous systems  
17 are not mature enough to fight off the toxins which are in the  
18 environment.

19 In the brain, because of its complexity the neurological and  
20 neuroimmune mechanisms, a comprehensive yet focused approach to the  
21 core disease mechanisms is necessary. Further the potential for a  
22 very insidious onset of signs and symptoms due to the\_ slow  
23 accumulation of damage makes sophisticated testing and pathological  
24 analysis essential.

25 However, as highlighted by this invention some of the basic  
26 pathological mechanisms are now understood, and the treatment  
27 approach follows naturally from the pathology. One central  
28 mechanism is the condition wherein Oxygen does not sufficiently

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diffuse from the arterial blood to neurons which need Oxygen to transform Carbon Monoxide into more harmless Carbon Dioxide.

Another of the most central of these mechanisms is called excitotoxicity, which is due to breakdown of control over flow of Calcium ions into neurons. This tendency toward excitotoxicity makes the brain and spinal cord vulnerable to process such as reactivation of intra neuronal neurotropic viruses which leads to intracellular damage through mechanisms which are at present unspecified, but much inflammatory damage in the neuron is mediated by Calcium. It is clear that the excessive influx of Calcium into the neuron up regulates a host of secondary messengers that create a number of inflammatory problems. (Levitan, 1991)

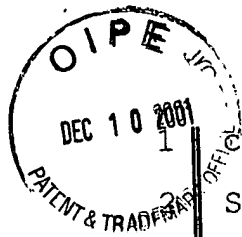
It was first noticed that Calcium had a special role in the transmission of information in 1977. (Fleckenstein, 1977) This was first noticed in ALS, where antibodies against L-Calcium channels were found. (Adams, 1993) This process has been called excitotoxicity. However, there is a second set of problems that arises with this type of damage called oxidative stress, from a combination of diminished availability of Oxygen, reduced ability to use Oxygen to make critical energy storage and biochemical molecules which participate in defending healthy tissue. "Free Radicals", which are abnormally electrically charged molecules are one of the results.

This type of metabolic and neuroimmune stress causes a number of problems to the central nervous system, the liver, kidneys, and to the immune system itself. For the nervous system oxidative stress appears to cause damage to the acetylcholine receptors, especially the nicotinic receptors associated with motivation,



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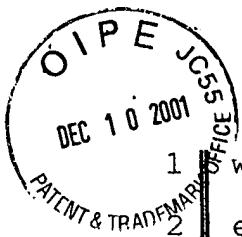


1 concentration and mental control. (Baumzweiger National Academy of  
2 Science Presentation to the institute of Medicine, October 16,  
3 1999)

4       The combination of these two problems of toxicity and  
5 infectious disease, which play off against each other makes many  
6 processes in the nervous system appear to be able to reactivate  
7 sequestered live virus, fungus and bacteria. When these organisms  
8 are captured and sequestered by healthy brain cells, they are  
9 generally stopped from reproducing and re-infecting neighboring  
10 cells. When they break out, as has been recently demonstrated in  
11 multiple sclerosis, there is reactivation of virus with  
12 inflammatory destruction.

13       Through advanced Lymphocyte testing, viral antibody testing,  
14 fungal antibody testing, venous partial pressure of oxygen tests,  
15 and testing for autoimmunity, there appears to be a condition  
16 localized to the brain stem and limbic system of the brain (BALD).  
17 Further there is a tendency towards BALD neurodysimmunity which is  
18 a unique mixture of immune suppression and multi system  
19 autoimmunity which is not seen in any other disease entity.

20       There have been numerous attempts to localize dysfunction or  
21 pathology to specific areas of the brain. For example John in US  
22 Patent 6,052,619 discloses a brain function scan function. This  
23 system is for purposes of differentiating between diagnosis of so  
24 called cerebrovascular obstruction (stroke), injury to the brain,  
25 injury to the spinal cord, or simply hemorrhage. John points out  
26 that these conditions need to be differentiated in order to  
27 properly treat the patient. John also points out that cardiac  
28 symptoms can be caused by a brain tumor which could be discovered

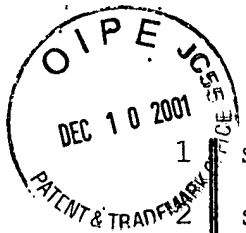


1 with his invention. This system is based on the so called EEG or  
2 electroencephalograph. While it is true that the EEG can detect  
3 brain tumors and other damage to the brain the system of John fails  
4 to recognize that it is possible to localize a disorder to the  
5 brain stem and limbic system itself. Nor does the John invention  
6 disclose a procedure to localize a disorder to the brain stem and  
7 limbic system of the nervous system.

8 Another relevant invention is the disclosure of Don in US  
9 Patent 6,080,112. This invention is a procedure to detect tumors  
10 in the brain using what is called the auditory brainstem response  
11 or ABR. This procedure would detect small tumors in the brain.  
12 However, the procedure does not go beyond the detection of tumors  
13 in the brain. This procedure uses the response to sound of one of  
14 the twelve cranial nerves the vestibulocochlear nerve and its  
15 cochlear branch. The cochlear branch of cranial nerve VIII conveys  
16 impulses associated with hearing or sound to the brain. This  
17 invention would not, however, localize a disorder to the brain stem  
18 and limbic system because for one thing it is not conclusive of  
19 brain stem damage. Cranial nerve VII damage could be caused by  
20 damage to the inner ear or other parts of the cranium and brain  
21 than the brain stem and limbic system. Also Don fails to recognize  
22 that if there is a disorder of the brain stem and limbic system  
23 taken as an entity then there would be many other symptoms than  
24 simply a possible disorder of one of the cranial nerves.

#### 25 SUMMARY OF THE INVENTION

26 The history of medicine is filled with flawed or failed  
27 attempts to localize a disorder to a specific area of the brain  
28 except for detection of brain tumors, vascular accidents called



1 strokes or trauma to the brain. This is due to the central nervous  
2 system being a complex system whose individual parts are closely  
3 inter-related so that a disorder of one part of the central nervous  
4 system could be caused by still another and related part of the  
5 CNS.

6       There has been a long felt need for diagnosis and treatment of  
7 the above described problems of neuro toxicity. Because the  
8 problems involved in localizing pathology to separate areas of the  
9 central nervous system the signs and symptoms of the present  
10 invention are subtle and often have been dismissed or overlooked by  
11 the physician. Thus, one principal object of the present invention  
12 is to delineate the clinical signs and symptoms and laboratory  
13 findings resulting from prior toxic exposure perhaps worsened by  
14 specific toxic substances, trauma, or infectious as well as  
15 autoimmune disease. This will result in changes in medical  
16 practice, with a focus on the pathologies due to toxic substances  
17 in the modern environment.

18       Because it is little recognized that the nervous system can  
19 and does react with repeated combinations of toxic exposure and  
20 infectious disease, and tissue damage, even when the individual  
21 insults are sub threshold A protocol for the diagnosis and  
22 treatment of the resulting clinical syndrome and damage to specific  
23 areas of the deep brain systems is desirable. With this invention,  
24 it will be possible to assess and treat the damage and the  
25 vulnerability to further damage from such combinations, as well as  
26 their complications such as reactivation of neurotropic virus, and  
27 subsequent excitotoxic damage. (Brewer, 1998) A similar tendency  
28 toward complexity can be seen in the newly elucidated genetic

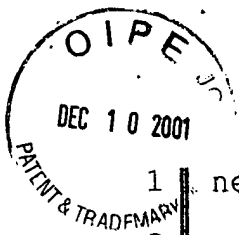


1 etiology of schizophrenia and the participation Chlamadia appears  
2 to play in heart disease, and which Helicobacter plays in the  
3 patho-genesis of peptic ulcers.

4 An extreme case of central nervous system dysfunction caused  
5 by toxic substance would be victims of so called nerve gas used in  
6 actual wartime environments. It is a principal object of the  
7 present invention to localize the central nervous system disorder  
8 that is caused by such toxic substances and to describe a treatment  
9 protocol for the disorder.

10 Up until the advent of the present invention, it has not been  
11 possible to clinically localize BALD central nervous system  
12 disorders to where they originate, the brain stem, the basal  
13 ganglia connected to the brainstem, the thalamus, which coordinates  
14 the rest of the brain by referencing these deep structures, and the  
15 axonal transmission pathways to the rest of the brain from the  
16 brainstem, basal ganglia, and limbic system. Localization of CNS  
17 disorders to the brain stem and limbic system is another principal  
18 object of the present invention.

19 Many disorders related to the brainstem and limbic system have  
20 been mis-diagnosed in the past. A few, such as Parkinsons Disease  
21 and Acute Brainstem Encephalitis are well known, but the brainstem  
22 and limbic system area has not leant itself to classic techniques  
23 of neurological localization. It has had less attention than it  
24 deserves. Chronic damage to and inflammation of these areas of the  
25 CNS are still not accepted by many physicians although they are  
26 well known and accepted by specialists in this area. Further,  
27 damage to the brainstem and limbic system is hard to discern, and  
28 a very careful, systematic, and highly technical approach is



1 needed. It is the principal objective of the present invention to  
2 correct this situation.

3       The primary purpose of the present invention is to localize  
4 and discern the underlying mechanisms which are driving the chronic  
5 signs and symptoms, and then treat the resulting disorders. As with  
6 all illnesses, eliciting the relevant elements of medical history  
7 is essential. As with all illnesses, a history of infections which  
8 cause neurological symptoms, a history of nervous system trauma, a  
9 history of exposure to environmental, industrial, or wartime toxic  
10 or poisonous substances is also essential. This invention involves  
11 a diagnostic protocol involving the patient history then a clinical  
12 examination to localize the area of nerve tissue damage to the  
13 brain stem and limbic systems.

14       Corroborative laboratory tests and other tests can be run as  
15 well.

16       The damage which charged particles can do to tissues is  
17 typified by the Calcium ion entering neurons and other cells.  
18 Calcium ion is an important signaling ion in the nervous system.  
19 With excess of Calcium in the neurons too little learning occurs or  
20 proper response to the environment does not take place, and the  
21 subject becomes somnolent.

22       The present method of diagnosis of the BALD syndrome is aimed  
23 at reduction of brainstem and limbic system dysfunction by medical  
24 treatment in two critical ways. First it would reduce the damage  
25 from Calcium ion in the nervous system no matter what the cause.  
26 Secondly it would aim to repair the damage caused by prior  
27 excitotoxic damage to neurons, free radical damage to proteins, as  
28 well as inflammatory and infectious damage to critical bodily



1 tissues.

2       There are two general approaches to localization of a disorder  
3 to the brainstem and limbic system. The physician could start with  
4 the physical symptoms such as cardiac or pulmonary symptoms which  
5 would indicate possible damage to the brain stem. Another approach  
6 is to relate emotional or psychiatric symptoms to the limbic system  
7 then include recognition of associated dysfunction to the brain  
8 stem as well in the diagnosis. Clinically it has recently been  
9 found that a disorder of the brain stem invariably includes a  
10 related disorder to the limbic system and visa versa. Thus it is  
11 a principal object of the present invention to provide a protocol  
12 for diagnosis of physical damage to the brain stem and limbic  
13 system associated with psychiatric symptoms.

14       In the drawing:

15       Fig. 1 is a decision table for the diagnostic protocols for  
16 the BALD syndrome. This drawing shows the four main steps to  
17 diagnosis: patient history, clinical tests, laboratory tests, and  
18 diagnosis confirmation.

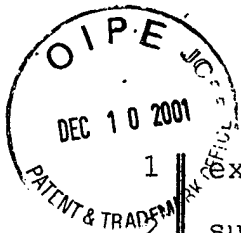
19               DETAILED DESCRIPTION OF THE INVENTION

20       This invention consists of a diagnosis protocol for  
21 localization for the first time in medical history to a disorder of  
22 the brain stem and limbic system.

23       The diagnostic protocol consists of 1) patient history; 2)  
24 clinical signs and symptoms 3) corroborative tests and procedures  
25 and finally 4) clinical confirmation by initial treatment of the  
26 disorder.

27       Patient history:

28       The key is obtaining a history of exposure or possible



1 exposure to environmental or industrial toxins or poisonous  
2 substances. Often there is the history of itching or burning of the  
3 scalp, shoulders or neck with possible neck and shoulder weakness  
4 which localizes dysfunction to the area of cervical segments C1  
5 through C4 inclusive. There is often a history of numbness,  
6 weakness, or discomfort from peripheral neuropathy.

7 Signs of the CNS aspects of the BALD disorder are photo phobia  
8 and headache. Often there is a history of cognitive deterioration,  
9 memory problems, and insomnia. The patient will often experience  
10 occasional dizziness on standing, difficulty with walking straight,  
11 dizziness, difficulty with swallowing, neck weakness, an odd taste  
12 in the tongue usually tinny or metallic. There is usually decreased  
13 smell insensitivity for normal smells, but over reaction to very  
14 strong smells, and sometimes even olfactory hallucinations. Often  
15 there are accompanying gastrointestinal symptoms.

16 There can be olfactory dysfunction such as over sensitivity to  
17 certain odors then loss of sensitivity to other odors.

18 Cranial nerve dysfunction can produce myriads of symptoms and  
19 this indication of possible BALD disorder should be used  
20 adjunctively in connection with other indications of the disorder  
21 from the patient history.

22 Symptoms of hypercalcemia include symptoms of lethargy,  
23 weakness, anorexia, nausea, vomiting, polyuria, itching, bone pain,  
24 depression, confusion, paresthesia, stupor, and even coma. Such  
25 symptoms clearly indicate the BALD syndrome.

26 Extra pyramidal system symptoms involve upper motor neurons  
27 and lower motor neurons. Mild extra pyramidal system symptoms  
28 could indicate the BALD disorder.



Memory dysfunction and emotional disorders are indicative of dysfunction of the limbic system. Clinical trials have recently shown that a disorder of the limbic system almost invariably involves a disorder of the brain stem as well.

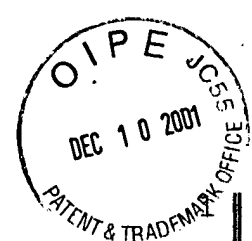
Clinical signs and symptoms:

Invariably the patient will experience abnormal increase in heart rate on standing, cranial nerve dysfunction, and in advanced cases mild extra pyramidal symptoms. The reflexes in the upper limbs are usually normal which distinguishes the disorder from other CNS disorders such as stroke and peripheral neuropathy. The reflexes and speed present at the knee are brisk, and other limb reflexes such as crossed adduction will be abnormal demonstrating the continued spreading of excess electrical excitability in the nervous system. Further, superficial pathological reflexes such as the glabellar, grasp reflex and finger flexion reflexion reflexes will appear. The normal infantile reflexes such as the tonic neck, placing reflex and crossed adductor reflex will re-appear. Multiple fasciculations appearing in muscles almost always signifies lower motor neuron dysfunction.

Step one in the clinical examination is to examine the sitting to standing heart rate. Usually this is not more than an increase in 10 to 15 beats per minute in the first few seconds after standing.

The stethoscope and a stop watch can be used or a pulse oximeter can be employed in this test. One check of the heart rate is made on sitting for 5 minutes, then after standing immediately a check is made at 5 seconds, a check at 15 seconds then a third check at 60 seconds. An abnormal increase on the





1 sitting to standing heart rate indicates dysfunction in the nucleus  
2 of Cranial nerve X of the brainstem .

3 The next step in the clinical examination is to examine one or  
4 more of the cranial nerves. Dysfunction in a plurality of the  
5 cranial nerves is a strong indication that there is damage to the  
6 brainstem.

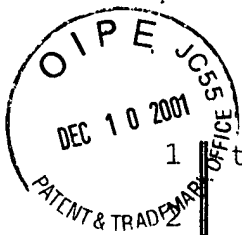
7 Examination of Cranial Nerve I the olfactory nerve:

8 With eyes closed the patient is asked to identify mildly  
9 aromatic substances such as vanilla, cologne or cloves. If there  
10 is a disorder of Cranial Nerve I this indicates damage to the  
11 anterior part of the brainstem.

12 Examination of cranial nerve II the optic nerve:

13 The peripheral vision test is used. The patient is instructed  
14 to look straight ahead. Then an object is brought into the  
15 peripheral vision of the patient and the patient asked to state  
16 when the peripheral object is first seen. Loss of peripheral acuity  
17 results from damage to the optic tracts for this retinal area, as  
18 they course directly over the inflamed parts of the brain,  
19 particularly the cingulate gyrus. In the BALD disorder the upper  
20 outer quadrants of the peripheral vision are morer affected than  
21 the lower outer quadrants. Paleness of the optic disk or edema of  
22 the optic disk is looked for. A patient with inflammation to the  
23 brainstem and limbic system, will often experience low tolerance or  
24 intolerance to light shown in the eyes. Many cases of photo-  
25 phobia are subtle. The patient must be asked very carefully about  
26 increased use of dark glasses and hats, or avoidance of the  
27 outside. This involves the clinical experience of the practitioner.

28 Examination of cranial nerves III, IV, and VI,, the oculomotor



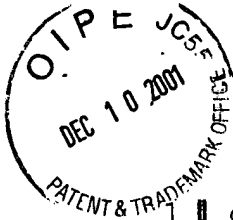
1 trochlear, and abducens nerves:

2       These three nenres are examined together, since they all act  
3 to control the extra ocular muscles. The patient is asked to blink  
4 as fast as possible. Fatigue of the levator muscles of the eyelids  
5 shows weakness in Cranial nerve III. A penlight is brought  
6 from a distance of several feet in front of the patient towards the  
7 eyes of the patient to test for visual convergence. The test is  
8 positive when there is diplopia up close. There can be observed by  
9 hyper convergence with double vision up close along with diplopia.

10       With lengthening of the penlight image distance horizontally,  
11 "sparkles" increase in the light of the penlight, or there is  
12 sudden darkening of the light or change in light color indicating  
13 cranial nerve dysfunction. These signs demonstrate loss of  
14 convergence at a distance . The light also is gradually moved  
15 backwards from the face, to twenty feet. At a distance a color  
16 change in the light can result. The most common color change is  
17 because of diffraction effects. At times the light will split into  
18 2 lights at a distance. At other times, sparkles will appear or the  
19 patient will note the light elongating horizontally. As with other  
20 cranial nerves we will describe, there is reduced dynamic range of  
21 the reflex control of the cranial nerve responses. This is seen in  
22 Cranial Nerves I, III, IV, V, VI, VII, and XII which all carry the  
23 special senses.

24 Examination of cranial nerve V, the trigeminal nerve:

25       This nerve carries sensory and motor neurons to specific areas  
26 of the face. The patient's facial sensation to pin prick,  
27 temperature, and vibration are tested. These are compared with the  
28 same sensations on the sternum. To test for damage to the third



1 division of this nerve the three areas of the face and neck  
2 enervated by this cranial nerve are tested and compared for  
3 sensation,.Sensory loss indicates damage to this nerve. Also small  
4 differences in sensation and delayed onset of sensory responses  
5 indicate damage to this nerve.

6 Examination of cranial nerve VII, the facial nerve.

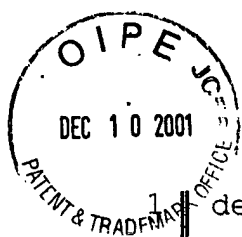
7 This nerve is tested for damage by asking the patient to  
8 perform various facial movements and checking for loss of sensation  
9 inside of the external ear. The pupil of the eye may be wide. There  
10 may be fewer wrinkles on one side of the face, and there may be  
11 asymmetry to voluntary smiling or forehead wrinkling. Weakness of  
12 the nerve can be detected also by asking the patient to detect  
13 sweet, salty, and sour substances that are applied to the tongue.  
14 Loss of taste sensation definitely would indicate damage to this  
15 nerve.

16 Examination of cranial nerve VIII the vestibulocochlear nerve.

17 This nerve transmits sound which allows a person to hear.  
18 Using a tuning fork the patient is asked to detect sound of lower  
19 frequencies from the tuning fork using air conduction and bone  
20 sound conduction. Loss of sound detection indicates damage to  
21 this nerve.

22 Examination of cranial nerves IX and X.

23 If cranial nerve IX is damaged the pharynx tonsillar pillars  
24 will show diminished sensitivity, especially on the side opposite  
25 of the greatest brainstem irritation. Cranial nerve X damage will  
26 cause a movement of the soft palate to the side, instead of the  
27 normal movement forward, when the tonsillar pillars are stimulated.  
28 Normally the soft palate should be symmetrical and should not



1 deviate to either side. When speaking "me, la, ka" from either side  
2 of the mouth, there may be a subtle deterioration of pronation,  
3 particularly on the side of the greatest brainstem irritation.  
4 Failure to properly enunciate these phonemes indicates damage to  
5 cranial nerve X. Further, these cranial nerves enervate the carotid  
6 sinus and the carotid bodies and damage to this nerve can be  
7 detected with the carotid body reflex which results in heart rate  
8 and blood pressure changes when moving from standing from a sitting  
9 position. The orthostatic tachycardia reflex which is reflex  
10 tachycardia resulting from change in position from lying to  
11 standing and which lasts only a few seconds can be used to detect  
12 damage to cranial nerve X. This reflex tachycardia can be best  
13 detected using a pulse oximeter. This cranial nerve controls the  
14 gastrointestinal tract, and gastroesophageal reflex so that  
15 constipation and diarrhea are very frequent in the BALD syndrome.  
16 Examination of cranial nerve XI, the accessory nerve.

17       This nerve is a motor nerve enervating the stemocleidomastoid  
18 muscle. To test this muscle for strength the patient is asked to  
19 turn the head toward one shoulder and to resist attempts of the  
20 examiner to move the head in the opposite direction. Then the test  
21 is repeated on the other side. Weakness in this muscle indicates  
22 damage to this cranial nerve. This nerve which moves the back of  
23 the head to the side being tested is the only cranial nerve with  
24 ipsilateral cortical connections.

25 Examination of cranial nerve XII, the hypoglossal nerve.

26       This nerve can be tested by asking the patient to push the  
27 tongue against either cheek then testing the strength of the tongue  
28 by pressing from the outside of the cheek. Fasciculation of the

1 tongue, involuntary movements with the tongue at rest can be seen  
2 in advanced disease,

3 Spinal cord Damage

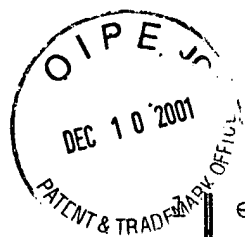
4       The next step in the clinical examination is to test for upper  
5 spinal nerve damage. The strength of the trapezius muscle can be  
6 tested by asking the patient to push the muscle against the hands  
7 of the examiner. This checks for damage to spinal segments C-3 and  
8 C-4. Any weakness in the neck muscles especially on flexion of the  
9 head indicates weakness of the nerve damage to the brain stem and  
10 high cervical cord. Due to viral infection there may be shingles,  
11 or loss of sensation on spinal dermatomes, or in some cases  
12 weakness below the high cervical spine. There may be decreased  
13 strength of respiration, with shallow breathing, and difficulty  
14 with ventilation.

15 Motor Examination.

16       The next step In the clinical examination is to check for  
17 increased motor tone by moving joints in the arms over their  
18 passive range of motion and checking for involuntary muscle  
19 resistance. Spasticity or loss of fine motor cc-ordination  
20 indicates upper motor neuron damage. Increased motor tone indicates  
21 upper motor neuron damage, possibly damage to the brainstem and  
22 other brain structures in close proximity to the brainstem and  
23 limbic system.

24 Peripheral Nerve Examination:

25       Peripheral nerve damage using the pin prick test and  
26 temperature test to check for loss of sensation is useful. A cool  
27 tuning fork is handy for checking the limbs, as well as the face  
28 for temperature. There is often loss of two point discrimination



especially on the side opposite the most affected part of the  
brainstem. In the advanced stages of the BALD disorder decreased  
sensation, altered sensation, and delay in experiencing sensation  
is observed.

In the BALD syndrome there is loss of stereognosis, so the  
patient cannot touch the area of skin the examiner has touched.  
There is, further, a loss of sensation or dyesthesia, abnormal,  
uncomfortable sensation on one or both sides of the limbs. There is  
loss of the ordinary ability to touch one's fingers together behind  
one's back with the eyes closed in the BALD syndrome. The normal  
person should be able to do this three times in a row without  
missing.

The next step is to check for abnormal reflexes, such as a  
mild glabellar reflex. Other superficial reflexes are usually  
normal.

There is usually no Babinski sign. With localized damage to  
one side of the brainstem and limbic system there may be a partial  
Babinsky on the opposite side that is damaged.

Reappearance of infantile Reflexes:

With brainstem damage infantile reflexes appear. The reflexes  
checked are: crossed extensor reflex, contra lateral reflex arc,  
deep tendon reflexes, the tonic neck reflex is checked for  
reappearance, and the infantile grasp reflex is checked for  
reappearance. The infantile placing reflex is checked for extension  
of the leg muscles upon rubbing of the shin.

Abnormal cardiac rhythms can be detected with the stethoscope  
and stop watch or with an Electrocardiograph examination if  
necessary.

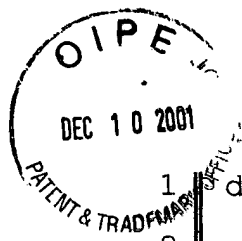


The clinical examination must check for lung and bronchial apparatus dysfunction. Prolonged expiration may be heard. The spirometry test is used and a number of these patients with brainstem and limbic system damage show mild restrictive airway disease. Increased auscultation usually points to increased intestinal motility.

Laboratory tests:

The next step in the diagnosis protocol is to run laboratory tests to corroborate brain stem and limbic system damage. With neuro toxicity and damage to neurons there will be abnormal levels of evidence of viral and fungal infection. Specific, tests for abnormal levels of virus presence such as the Barr-Epstein virus CMV virus, HHV6 virus, and HHV2 virus can show abnormal vulnerability to viral infections and re-infection. The T4/T8 lymphocyte levels can be tested for abnormality in the immune system. On the usual lymphocyte panel tests used to identify immune system dysfunction there will be evidence of both immune suppression as well as evidence of auto immunity in the BALD syndrome. The T(4)/T(8) cell ratio is either too high or too low. The test for NADH is abnormal indicating neurotoxicity and damaged neurons. In a nutshell abnormal results of tests for abnormal antibodies to neuron components reveals damaged neurons.

It is possible to run a coagulation panel or serum profile to be used to check for 1) fibrinogen antigen, 2) heparin assay, 3) thrombin/anti-thrombin complexes, 4) soluble fibrin monomer, and 5) platelet associated Ig G. Immune system activation of slow cold agglutination and increased free fibrin escaping in to the serum show immune system dysfunction and activation of coagulation,



1 demonstrating a cause for tissue asphyxia or hypoxia. The root  
2 cause of this immune system dysfunction is brainstem dysfunction.  
3 Further the root cause of abnormal blood clotting is excitability  
4 of neurons and blood vessels from excessive intake of Calcium ions  
5 into the neuronal cells and blood vessel cells.

6 The next step in the diagnostic protocol is to run a MRI check  
7 with a long T-2 sequence, using Gadolinium, on the brainstem and  
8 limbic system. Necrotic tissue and gliosis can be observed in this  
9 test in severe cases of the BALD syndrome.

10 Finally a brief neuropsychiatric examination should be done  
11 checking for 1) loss of the sense of the familiar, 2) recent  
12 onset of obsessive behavior, 3) loss of predictability in behavior,  
13 and 4) decreased interpersonal involvement. Emotional disorders may  
14 be present such as hysterical responses to certain events.

15 Confirmation of the diagnosis:

16 Usually the BALD disorder involves damage to the medulla  
17 cardiac center which regulates the heart rate. In the BALD  
18 syndrome the cardiac center of the medulla is damaged by excessive  
19 calcium ions in the neurons. This condition can be alleviated by  
20 administration of so called calcium intake channel blocking drugs.  
21 These drugs block intake of calcium ions to the neurons which would  
22 improve the function of the cardiac center. Administration of a  
23 calcium intake channel blocking drug which shows improvement in the  
24 sitting or prone position to standing heart rate change would serve  
25 to confirm the diagnosis of the BALD syndrome.

26 Excess calcium ions in the cells of the body has been  
27 clinically found to impair the function of the TCA cycle of the  
28 cells. Administration of the drug NAD would promote return to a





1 more normal TCA cycle in the cells of the body and would tend to  
2 confirm diagnosis of the BALD syndrome.

3 It should be emphasized that administration of a calcium  
4 intake channel blocking drug and/or NAD is not an effective  
5 treatment protocol of the BALD syndrome but can be effectively used  
6 to clinically confirm the diagnosis. A complete treatment protocol  
7 would then be indicated.

8 The above description of the diagnostic protocol of the BALD  
9 syndrome is for purposes of illustration and not for purposes of  
10 limitation. The limitations of the present invention are set forth  
11 in the claims